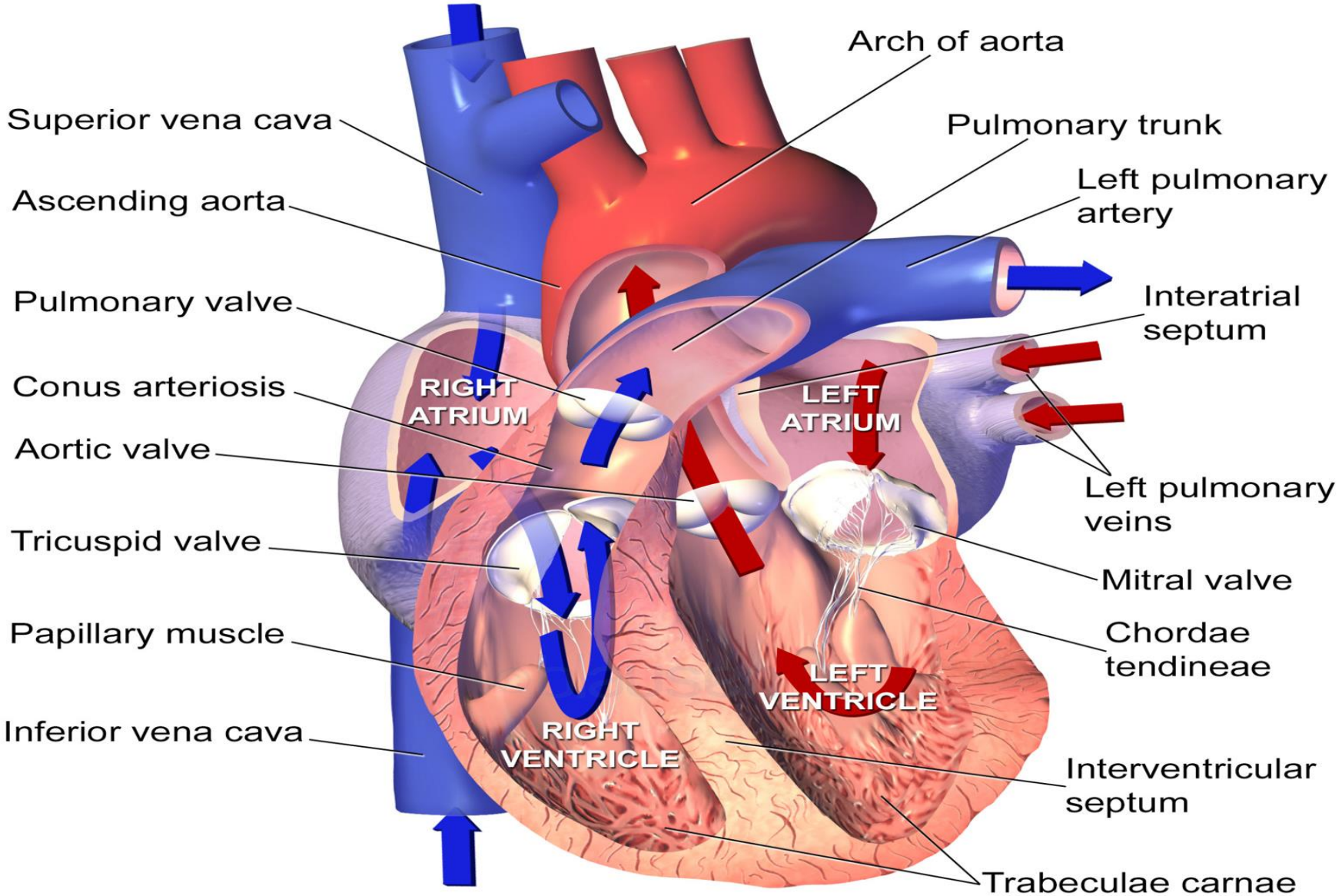


Sectional Anatomy of the Heart

Pathophysiology of CVS

- The heart functions like a pump
- with each beat it pushes blood into circulatory system
- It is composed of 4 chambers — 2 Atria — Right & left
  2 Ventricles " "
  both Rt. & lft chambers are seperated by a septum.
- Left side of the heart chamber is a strong pump which is responsible for pumping the blood to whole body.
- Normal heart weight varies b/w individual body weight & height.
  Avg wt 250 to 300 gm in females &
  300 to 350 gm in males.
- Greater the heart wt & thickness of Heart walls called "Hypertrophy"
  where as Enlarged Heart size is dilatation
  Rise in size & weight termed as cardiomegaly
  Usual thickness of walls of Rt ventricle is 0.3 to 0.5 cm
  & Lft ventricle: 1.3 to 1.5 cm

(2) Blood Supply : Coronary arteries which arise from aorta 5-10cm long & 2-4 mm diameter. Run along the surface of heart. Supplies oxygenated blood

Cellular waste & CO_2 from the body along & blood i.e Impure blood enters Rt atrium via Superior & Inferior Vena Cava

Finally pumped the Left Atrium Valve into aorta

In Lt ventricle

Through Mitral valve

Lt atrium

From pul veins

Oxygenated blood

From lungs CO_2 Expelled

(C.I Each breath)

Where it branches into arteries arterioles capillaries delivering oxygenated blood to tissues of body

From Rt atrium

↓

blood flows into Rt Ventricle

↓

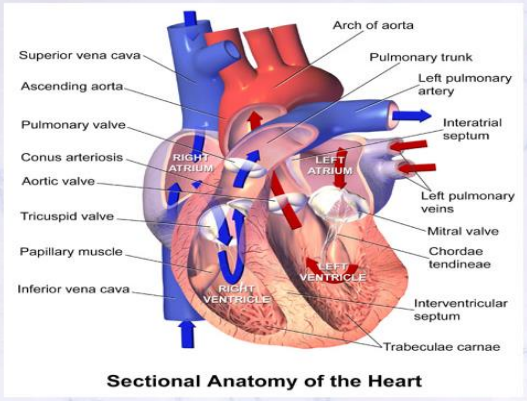
From pul artery (through pul valve)

↓

Into Lungs.

(Oxygenation occurs)

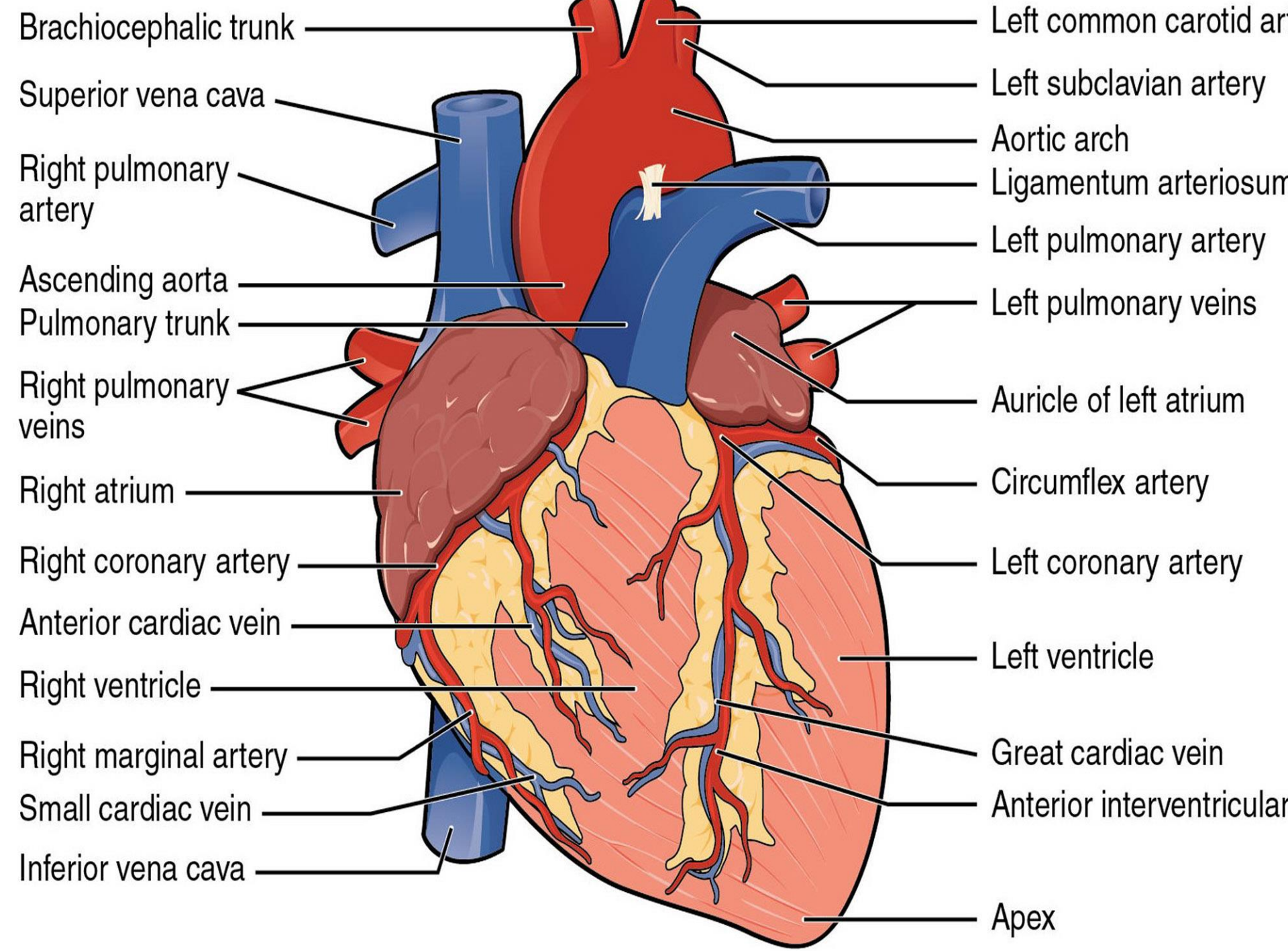
In Exchange for CO_2 which is * Expelled during Expiration



Sectional Anatomy of the Heart

— As blood brings oxygen & Nutrients to body tissues it also removes CO_2 & waste products

— This flows into venules to larger veins finally back to the right atrium wherein contains


Brachiocephalic trunk
Superior vena cava
Right pulmonary artery
Ascending aorta
Pulmonary trunk
Right pulmonary veins
Right atrium
Right coronary artery
Anterior cardiac vein
Right ventricle
Right marginal artery
Small cardiac vein
Inferior vena cava
Left common carotid ar
Left subclavian artery
Aortic arch
Ligamentum arteriosum
Left pulmonary artery
Left pulmonary veins
Auricle of left atrium
Circumflex artery
Left coronary artery
Left ventricle
Great cardiac vein
Anterior interventricular
Apex

Here comes Pathology

Diseases that affect heart called cardiac diseases:

There are Many diseases which affect heart & blood Vessels.

Results from one or more of 5 principal Mechanisms

① failure of the pump : i.e Heart

Cardiac Muscle becomes weak & it Cannot Empty the blood properly.

It Cannot relax to permit ventricular filling

leads to pump failure.

② Obstruction in the flow : 1. Defective valvular opening

2. Systemic Hypertension

will ↑se the pressure in the chamber so which ↑ses the workload on the heart causing Hypertrophy

(3) Regurgitation : Blood flowing backward with each contract
- on during cardiac output adds excess workload to each Chamber → where it has to pump Extra blood.

(4) Disorders of cardiac contraction : Due to
Uncoordinated generation of impulses in
Heart blocks & in arrhythmias causes
non uniform and inefficient contractions
of cardiac muscle

(5) Cardio Vascular disease also arise from
interaction of Environmental factors & genes susceptibility
which disrupt network & control morphogenesis -
Myocyte survival, biochemical stress responses
Contractility & Electrical conduction.

Disrupts the Network.

1) Myocyte survival
2) Biochemical stress respons
3) Contractility
4) Electrical Conduction

Pathophysiology : Problem arises when blood flow is used or blocked. Usually blocked by atheromatous plaques to the arterial walls. causing narrowing of vessel calibre thus reducing flow of blood to heart.

So the Heart does not get enough O_2 & Heart attack results.

If the vessels that feeds brain is blocked results Stroke

Resulting Millions of deaths due to cardio vascular : causing death

As the heart is damaged due to insufficient O_2 it becomes less Efficient

↓

And has to work Much more

↓

To Deliver sufficient O_2 throughout the body

As the time passes Heart disease leads to Major problem

involving Lungs
Kidneys
* Eventually Every system of body as Every organ depends on heart for O2 & Nutrient supply.

Commonest cardiovascular disease is

1. Hypertension : (HTN)
2. Congestive Heart failure (HF)
3. Ischemic Heart disease. (IHD)
4. Atherosclerosis
5. Arteriosclerosis
6. Aorhythmias

Hypertension : Def : High Blood pressure.
BP is the force which the blood put against the wall of arteries as it flows through them :
(Here the arteries are the vessels which carry Oxygenated blood from Heart to the body tissues) BP = Cardiac output × peripheral resistance

The higher the Blood pressure
The Blood Exerts the pressure on artery walls.
When Muscular walls of arteries relax or dilated
Pressure is lowers than the constriction of artery wall.

When pressure is Measured
Systolic is recorded first
and than the Diastolic.
Measured as mm of Hg.
Ex: Sys pressure 120
Diastolic 80.
written as 120/80 mm of Hg.

Hypertention ~~tends to~~
is High risk. which leads to heart disease.
Regular checking of B.P & treatment as soon
as it is diagnosed avoid serious complications

If left untreated hypertension leads to
- Atherosclerosis
- Heart attack
- Stroke
- Enlarged heart
- Kidney damage.

High blood pressure makes the arterial walls thickened and harden and makes blood vessels narrow as cholesterol & fats deposit on the walls of damaged arteries & makes narrower.

Blood clots get trapped in narrowed arteries blocking the flow of blood.

Narrowed vessels does not deliver Enough blood to organs & tissues
↓
Cause Heart attack, Stroke.

In kidneys: Where its function is to remove waste from blood by filtration
Due to Hypertension of arteries of kidney thickens & filter less waste & waste builds up in the blood. So Dialysis or Kidney transplant

needed of when kedney fails

Epidemiology: → which deals ē Incidence Distribution g Diseases.

According to world Health Statistics } - In 2012

Hypertension Prevalence: 29.2% in males
24.8% in females.

out g Total 58.8 million Deaths world wide in yr 2004
Deaths due to HT2 was : 12.8% i.e 7.5 Million deaths.

CVA due to HT2 : 51%
IHA " " : 45%

Abont 33% Urban
25% Rural } Indians are Hypertensive -ve

25% Rural
& 2% Urban } Indians all aware g Hypertensive status

1/10th g Rural 1/5 g Urban Indian Hypertensive -ve population have their Bp under Control.

Etiology : i.e causes.

Hypertension : Due to physical activity or stressful conditions.

HTN :

Without known cause is called primary or Essential HTN

Secondary HTN : when caused by Medical condition

- Disorders of Kidneys
- Cushing's syndrome
- Tumors of pituitary & Adrenal glands (cortisol, Adrenalin, Aldosterone causes HTN).
- Thyroid gland disorders
- Alcoholism
- some prescribed drugs
- Pregnancy.

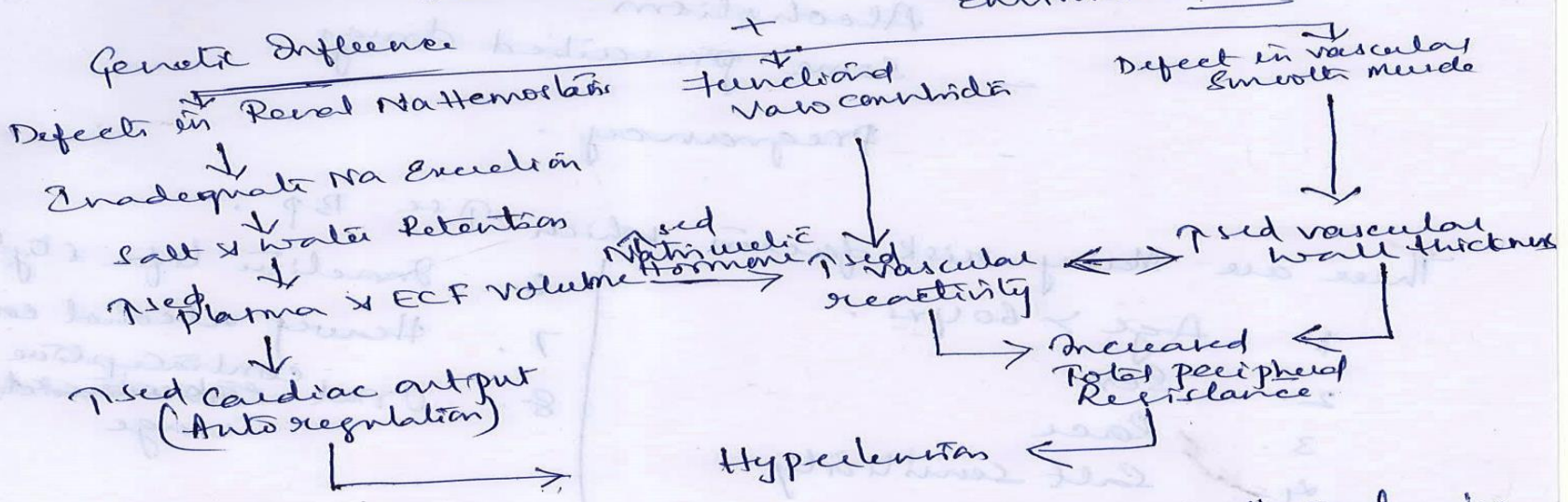
There are Many risk factors which Inc BP.

1. Age > 60 yrs.
2. Sex
3. Race
4. Salt sensitivity
5. Obesity
6. Inactive life style
7. Heavy alcohol consumption
8. Oral contraceptive usage

Genetic defects in renal Excretion of Na
functional regulation of vascular tone
Structural regulation of vascular caliber.

Environmental factors like
↑ed Salt intake, Potentiates
the Effects of genetic factors.

↓
Results in ↑se cardiac output
and peripheral vascular Resistance contribute
to HT2



Hypothetical scheme for Pathogenesis of Essential Hypertension

Natriuretic Hormones → released by Atrial & Brain (12) due to stretch Ventricular Natriuretic Peptide

Causes → Natriuresis.
→ Excretion of Na in the urine

Blood pressure Regulation

There are Multiple factors which influence the function of cardiac output and peripheral Vascular resistance.

Factors affecting cardiac output are
1. Sodium intake
2. Renal function
3. Mineralocorticoids

- The inotropic effects occur via Extracellular fluid Volume and an ↑se in heart rate & contractility
- Peripheral Vascular resistance depends on
  Sympathetic Neuronal system
  Hormonal factors
  Local auto regulation

Sympathetic Neuronal system produces its effects via
α effect or its vasodilator beta effects.
Hormonal actions (Kinins) or Vasodilators PR are Mediated by Vasoconstrictors Prostaglandins Kinins AT & Catecholamines

Autoregulation of BP occurs by way of Intravascular Volume contraction & Expansion as well as by transfer of transcapillary fluid

Interaction b/w Cardiac output & Peripheral resistance are autoregulated

For Example: constriction of arterioles elevates arterial pressure by ↑ing Total peripheral resistance.

But- venular constriction leads to redistribution of Peripheral intravascular volume to the central circulation, there by ↑ing preload & Cardiac output.

Preload : LVEDP (Left ventricular End diastolic pressure)
Amount of ventricular stretch at the End of diastole
As the heart loading upon next beat

After load : Known as Systemic vascular resistance
Amount of resistance the Heart must overcome to open the aortic Valve & push the blood into Systemic Circulation

Kidneys play an important role in Hypertension by 3 Mechanisms

1) Renin Angiotensin system :—

2. Sodium Homeostatis
3. Renal vasodepressor substances.

1. Renin Angiotensin System:

When renal blood flow is reduced
↓
Juxtra glomerular cells in Kidneys
Convert precursor (prorenin) already present in blood
↓
To renin
which converts Angiotensinogen released by liver
↓
Angiotensin I
↓ ACE (lungs)
Angiotensin II
↓
Is potent vasoconstrictive
Causes blood vessels to Narrow
↑ed B.P
Angiotensin II also stimulates Aldosterone from adrenal cortex
↑se Reabsorption of sodiums and water into the blood

Many drugs that intercept different steps in the System to lower Blood pressure.

2. Sodium Homeostasis : GFR & GFR independent.
Natriuretic factors play imp role in Sodium Homeostasis.

When blood volume reduced
↓
GFR ~~↓~~ falls
↓
Leads to ↑se reabsorption of Na by proximal tubules
to conserve Sodium & Expand blood volume

Natriuretic factors Secreted by Heart chiefly
in response to volume Expansion which
inhibits Na reabsorption at distal End.

3. Renal Vasodepressor Substances :

Prostaglandin, Urinary Kallikrein - Kinin system
platelet activating factor & Nitrous oxide.
Antihypertensive substances which counterbalance the vasopressor Effects of angiotensin.

Pathogenesis of Hypertension :

Pathogenesis of Essential Hypertension is multifactorial and highly complex.

Multiple factors affects the blood pressure

They include
1. Hormonal Mediators
2. Vascular reactivity
3. Circulating blood volume
4. Vascular caliber
5. blood viscosity
6. Cardiac output
7. Blood vessel elasticity
8. Neural Stimulation
9. Genetics
10. Excess dietary salt intake

Vasoreactivity of vascular bed is an important phenomenon Mediating changes of HTN

Activity of Vasoactive factors
↓ causes
Reactivity of smooth Muscle cells
↓ leading
Structural Changes in vessel wall & Vessel caliber
(Expressed by Lumen to wall Ratio)

Role of Vascular Endothelium : It is considered as

Vital organ

Where Synthesis of Various Vasodilators & constrictors occurs,

They include Angiotensin II → Potent Vasoconstrictor Synthesized from Ango I by ACE

Bradykinin

Endothelin → Potent Vasoconstrictor

NO → Play a Major role in Pathogenesis of HTN

↓

Extremely potent vasodilator that influences Local autoregulation

14.7a

Genetic factors : HTN Related to Multiple genes

The Exact Mechanisms that not Established

Hypertension Summary

follows in Next page No 18

Risk factors

1. Family History
2. Age
3. High salt intake
4. Low potassium intake
5. Obesity
6. Excess Alcohol consumption
7. Smoking
8. Stress

↓

Changes in Arteriolar (Arteriolar bed).
↑ Systemic Vascular Resistance

↓

↑ Afterload

↓

↓ Blood flow to organs

↓

Blood pressure

↓ Renal perfusion.

Beta receptor activation

Hypovolemia

Juxtra glomerular cells

↓

Renin

Angiotensinogen

Angiotensin - converting Enzyme

Angiotensin I

↓

Angiotensin II

↓

Arteriolar vasoconstriction

↓

Peripheral Vascular Resistance

↓

Blood pressure

Adrenal cortex stimulation

↓

Aldosterone

↓

Na reabsorption

H2O reabsorption

↓

Plasma volume (ECF)

Δ^{bp} : Usually based on 2 or more readings

Normal pressure is 120/80 mm of Hg

Mild : 129/89 mm of Hg

Moderate : 140–159 / 90 – 99 mm of Hg

Severe : 160 – 179 / 100 – 109 mm of Hg or above.

Physical Examination includes

1. Medical History
2. family history
3. Ophthalmoscopy → Thickening, Narrowing Haages of vessels.
4. Chest x ray → Detect Enlarged Heart-
5. ECG → If Muscle damaged or Enlarged
6. Blood & urine test → Detect presence of Disorder of HTN

Several blood pressure readings at different times & in different positions required to diagnose.

Treatment: No cure for primary Hypertension.

Secondary: Treat the cause which is responsible

Treatment in combination ① Medications
and ② life style changes.
③ Low salt diet
④ Reducing fat intake
⑤ Reducing wt.
⑥ quitting smoking
⑦ Reducing alcohol consumption
⑧ Managing stress.

Antihypertensives: 1. Diuretics
2. Beta blockers
3. Calcium channel blockers
4. ACE Inhibitors
5. A α blockers
~~6. β blockers.~~
⑥. Vasodilators

1. Diuretics: Most commonly used drug. It is Drug of choice used as Part of any Multi drug combination

2. Beta blockers: Lowers blood pressure by acting on β receptors in blood vessels & heart & Slows down the Heart rate & reduce force of contractions.

3. Ca channel blockers : As Entry of Ca^+ into artery Muscle Cellwalls causes contraction → Hence blockers will prevent Entry & keeps them relaxed & lowers pressure.

4. ACE inhibitors : block the conversion of Angiotensin II from Angiotensinogen ↓ which is Powerful Vasoconstrictor

5. α blockers : Act on nervous system to dilate arteries

6. Vasodilators : Act directly on artery walls to relax & Lowering the blood pressure rapidly

Used in Hypertensive Emergencies when pt's have dangerously High pressures.

Prevention :

1. Indulge in physical Activity
2. Get More Sleep
3. Keep a Stress Diary
4. Talk to Someone
5. Listen to Music, Yoga, Meditation

By reducing Na intake → Excess of Dietary Salt
" fat intake
losing weight
quitting regular Exercise.
quitting Smoking
Reducing alcohol consumption.
Managing Stress.

Ischemic Heart diseases

IHD : It is Imbalance b/w Myocardial O_2 supply and demand

↓

Resulting Myocardial Hypoxia

Ischemia : Insufficient blood supply to an organ

Myocardial Ischemia : A condition of coronary artery blood supply is blocked leading to starvation of O_2

When it is completely blocked.

↓ Lead to

Heart attack (Infarction)

Death of that Particular tissue

Ischemia May be

- Silent → without any symptoms
- or Symptomatic → i.e chest pain, Vomiting, Sweating → called "Angina" or "Peclonis"

Types of Ischemic heart disease

- Myocardial infarction (Infarct effect)
- 1. Ch. Ischemic Heart disease
- 2. Sudden cardiac death
- 3. Angina pectoris
- (Non infarct effect)

Etiology: Causes of Ischemia caused by blockage of an artery due to plaque, ~~Blood clots~~

Silent Ischemia: Caused by Emotional/Mental stress Exertion

→ No symptoms.

Angina: Caused by ↑ed demand of O_2 when Heart is working more than usual during physical stress / during Exercise.

TIA: Caused by blood clot due to blockage of cerebral artery

Due to temporary deficiency of blood supply to the brain occurs suddenly and lasts for few min to few hrs.

It is strong warning signal of a stroke.

Risk factors

1. Heredity: Children born to parents having coronary artery disease → More likely to develop.
2. Sex: Men are more likely than women.
3. Age: > 45 yrs or older — Men } at Risk.
   55 yrs or older → women }
4. Smoking: ↑sed chances to develop.
5. High Cholesterol: ↑sed Risk ∝ high cholesterol.
6. High blood pressure: which Makes Heart to work More ∝ time becomes weak and along ∝ obesity, Smoking diabetes ∝ high cholesterol. ↑ses the risk of Heart attack
7. Lack of Physical activity: ↑ses risk of coronary artery disease
8. D M: Risk is ↑sed
9. Obesity: ↑ses strain on heart ↑ses Blood pressure ∝ cholesterol
10. Stress ∝ anger: ↑ses the risk of CAD.

Angina symptoms include: Tight squeezing
Heavy
burning
Pain felt beneath the ~~breast~~
Strainen spread
to neck, arm & back

↑ IA symptoms: Sudden weakness
Tingling or numbness in leg or arm on same side of body
Sometimes to face
Difficulty in speaking
Vertigo & loss of balance.

Diagnosis: ECG
Coronary angiography
Echocardiogram → Heart sound wave are converted into an image in monitor.
CT scan (computed tomography) ← Thin x ray beam shows 3 dimensional views of soft tissue
MRI (Magnetic resonance imaging)
PET (Position Emission Tomography)
↓
Scanner ~~face~~ provides 3 dimensional images of Heart tissue
Very Expensive — Rarely used.

Treatment : ⊂ Drugs
↘
Surgery.

Nitrates
Beta blockers
Ca channel blockers
} Relieve Chest pain but not block.

Block is only relieved by surgical procedures like Coronary angioplasty (PTCA)
Bypass graft surgery. (CABG)

Prevention : ⊂ Healthy lifestyle → proper Exercise & food
Maintaining healthy weight
quitting Smoking
Drinking less
Controlling HTN
Managing stress

By all these above measures we can reduce the risk of Ischemia progressing to Heart attack or stroke.

Myocardial infarction : Heart Attack

Word Myocardial because Heart Muscle changes that occur due to sudden deprivation of O_2 to Myocardium causing Necrosis

Infarction : word comes from Latin Infarcire Meaning to plug up or stuff → Refers to clogging of artery.

MI → Abbreviated form.

It is accompanied by severe chest pain
Sweating
Vomiting.

Epidemiology : Leading cause of death in US & in Industrialized Nations

4,50,000 People in US die from coronary disease — Per year

Survival rate : 95% — US

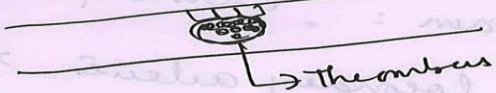
Pathophysiology : Etiology

Most common cause of MI is Narrowing of Epicardial blood vessel due to atheromatous plaque

1. 

2. plaque plaque gets ruptured Exposing base of vessel

3. platelet aggregation forming thrombus



Results Partial or Complete occlusion of vessel

4. If occlusion of vessel continues for More than 4–6 hrs

Results

Irreversible Myocardial Necrosis

When reperfusion is allowed within this period we can reduce the Morbidity & Mortality.

Other causes

Non atherosclerotic

Patient using – cocaine
– amphetamines
– Coronary Emboli due to infected heart valve
– Occluding coronary due to Vasculitis
– Chest trauma in Accidents & sports injuries

Diagnosis 1) ECG : Elevation of ST segment
T wave inversion
Q wave changes

2). Angiogram : catheter is introduced into coronary arteries & dye will be injected to Notice where the block is ... or ~~How much~~ what % of block is seen.

3. Biochemical parameters → creatinine kinase Cardiac bromarker Troponin (Muscle leak from the muscle when damaged)

4). Physical Examination :

Treatment : O_2 (Demand for O_2) Aspirin (Anti platelet activity) Nitroglycerine (Vasodilator) Analgesics (Morphine) Pain killed & sedate

administered as soon as possible

Reperfusion: By Thrombolytic drugs
Streptokinase
Urokinase
Heparin alone is ineffective as anticoagulant.
Aspirin to be added as standard therapy
Coronary bypass surgery is another option.

Angina pectoris: Heavy crushing pain in chest behind Sternum Mostly towards left.
Pain radiate to Lt arm, Left side of Neck & Shoulder blade.
occurs due to O_2 supply & other Nutrients are inadequate for Metabolic Needs of Heart Muscle.

Types of Angina
① Stable → occurs during activity or Exertion Quickly reversed by rest or taking Nitroglycerine Lasts for 3 - 20 min They are at risk of having Heart attack
② Unstable
③ Prinzmetal's

(2) Unstable : Where Heart does not get blood supply due to blockages in arteries

It occurs even at rest

(3) Prinzmetal's - Due to coronary vasospasm but not by plaque depositions

Occurs at rest or at odd timings at Night

More Common in Women < 50 yrs

More common in men than women c Mg insufficiency

Imp cause of MI

Etiology :

- Psychological Stress
- Physical Exercise
- Extreme cold
- Heavy meal

} Main Causes.

Angina can be aggravated by

1. Sustained fast heart beat
2. Anaemia
3. Heart valve diseases → Severe aortic Stenosis.
4. ~~Narrowing of Outflow~~
5. Thickening of Heart Muscle

Pathophysiology :

Atherosclerosis

Leads to Coronary Thrombosis

Leads acute coronary syndrome like unstable angina or MI

Number of attacks can be reduced by restoring its blood supply Either by balloon dilatation or bypass surgery

Angina pectoris

Narrowing of coronary arteries

↓

Insufficient blood flow

↓

Myocardial O_2 demands Exceeds Supply

↓

Anaerobic Metabolism & lactic acid accumulation

↓

Myocardial Nerve fibres Irritated

↓

Pain Message transmitted to Cardiac Nerve & upper posterior Nerve roots.

Systematic representation of process of angina pectoris due to blocked artery

Diagnosis: Based on presence of symptoms

Medical History: Whether GTN spray or tablet under tongue relieves pain.

ECG

Treadmill → Has to be done to determine

Treatment: 1. GTN Sublingual or spray → Relax arteries

2. Long acting Nitrates → Reduce frequency of attacks

3. Beta blockers: ↓ses Blood pressure & Slow down the pulse and reduces the Hearts need for O_2 & improve blood supply.

4. Ca channel blockers: Reduce Muscle tension in coronary arteries Expanding & creating more room

5. potassium channel activator: Also ↓ses tension present in vessel wall & Expands them improving flow of blood & O_2 supply.

Congestive Heart failure

Definition: Heart loose the ability to pump

Enough blood to the tissues

↓ leads to

Deficient O_2 & Nutrients supply to function properly.

Heart failure Mainly due to abnormality of left ventricle:

Left ventricle cannot pump as fast as it gets receives from the lungs

↓ causing

Back up of blood in blood vessels of lungs.

So some of the fluid from backup blood

Enters into breathing space of lungs

Causing pulmonary Edema

↓ Resulting

Shortness of Breath ⇄ Acute
Severe
↘ Life threatening

Rt sided Heart failure:

Rt Ventricle cannot pump enough blood to lungs as fast as it gets from the body through veins.

↓

So the fluid which is backed up forced into tissues causing (edema) swelling of feet.

Epidemiology

In India Ranges from 1.3 to 4.6 million

In 2000, 30 million people ∈ coronary artery disease in India alone OR 3% prevalence

8, 14 → Annual Incidence of HF ∈ CHD ranges from 0.4% to 2.3% Per year

15, 16 → Suggested 1,20,000 – 6,90,000 could develop ~~CHD~~ HF due to CHD Every year

Etiology

1. Coronary Artery disease
2. Heart attack (Silent)
3. Cardiomyopathy
4. High Blood Pressure
5. Heart valve disease.
6. Congenital Heart disease.
7. Alcoholism
8. Drug abuses

CAD : common cause of HF.

Arteries ⇒ Narrowed → Blocked
↓
flow ↓ed to heart
↓
Heart attack ← damage affected Heart
↓
Heart ability to pump fails

Cardiomyopathies — Caused by CAD * Various other Heart problem.
↓
Makes the Heart weak * Myocardial cells die ↓ replaced by collagen.
* leads to failure ↓ (Lost ventricular compliance)

High blood press : Where Heart works more to pump blood against peripheral resistance with the passage of time Heart cannot keep up ↓ leads to failure.

Pathophysiology : Reduced Cardiac output ⇒

2 Parts in pumping action) he to: ① Systolic Dysfunction
1 Part: Diastole — Blood collects in lower heart (Rt & ven)
↓
Because diminished Ventricular

Contractility ↘ Remodeling

Impaired stroke volume to meet demands

Once they are filled (4) 2nd part of pumping action begins. Ventricle contracts & pushes the blood from Rt to Pul art & Lt vent to Aorta (5) Systole

Few factors like (1) anatomical valvular obstruction

(2) ↑sed Peripheral vascular resistance

↓ lead to

↑sed Systolic Dysfunction

(2) Diastolic Dysfunction

↓ Remodelling from

↓sed Ventricular compliance

Do not relax, become stiff cannot fill blood properly

At the same time → Blood tries to Enter into ventricles from Next heart beat ↑ses the press in ventricles

↓↑

Then Exha press. Causes Pulmonary congestion in Lungs.

↓ Causes fluid leak into lung alveoli causing Pul Edema.

Elevation pressure in the vessels
that lead back to the heart
Reflected as systemic congestion

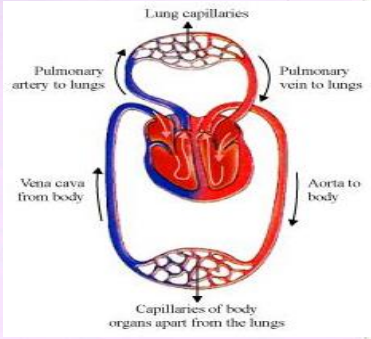
↓

Systemic congestion has effects on

↓

Kidneys & liver
↓ Renal ↓
Swelling & congestion
in legs & Abdomen

Pic of Blood Circulation 4B
both Pul & systemic Circulation



Causes of Diastolic dysfunction:

1) Anatomical valvular obstruction
2) Cardiomyopathy → ↓ Reduction in Ventricular compliance
3) External constraint → Pericardial effusion
4) Elevated pul vas resistance

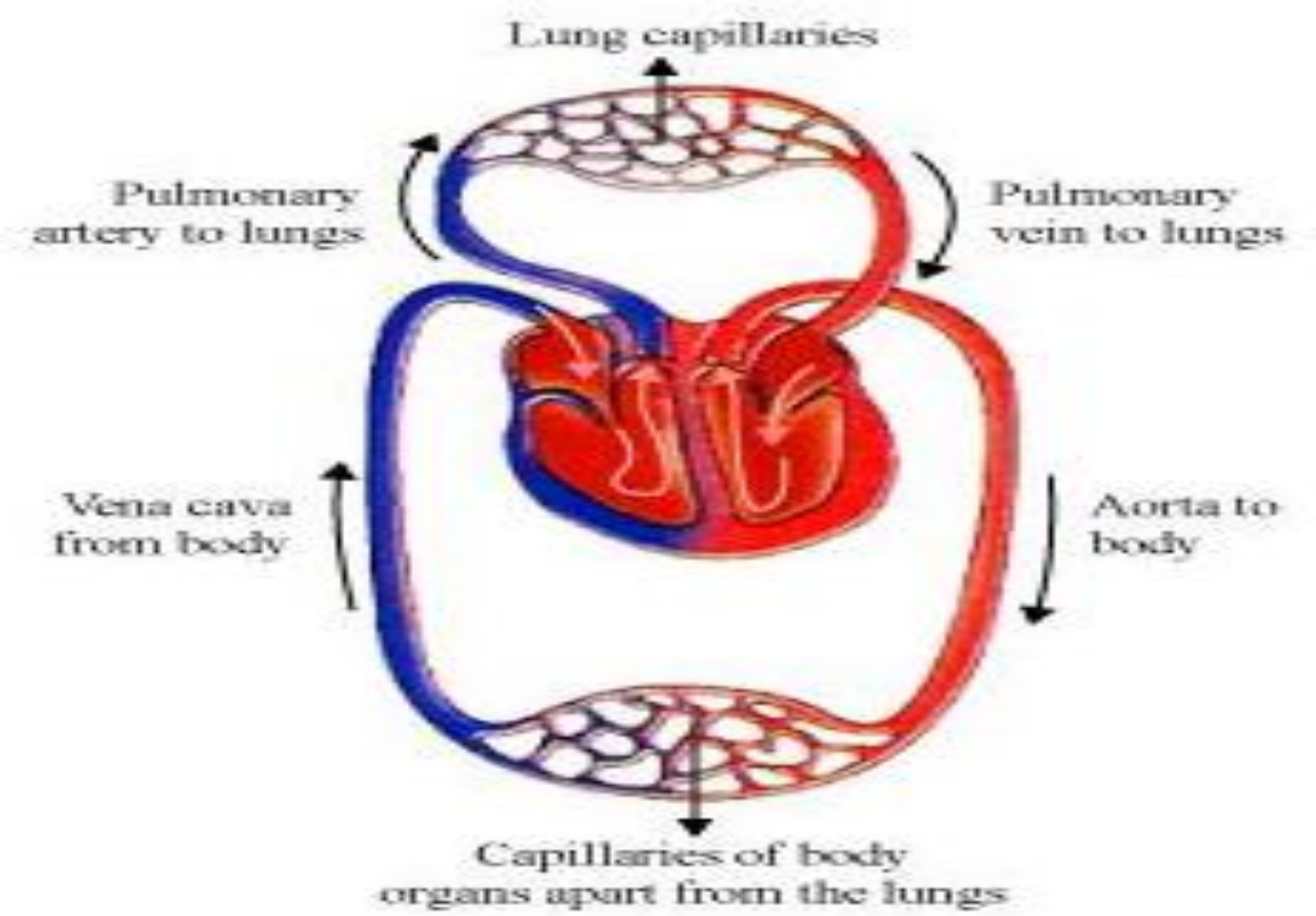
In Ch. Heart failure, Myocardial cells die due to Energy starvation, (Due to Mitochondrial loss) & lead to Necrosis due to cytotoxic Mechanisms.

↓ Replaced by collagen (As Necrosis stimulates ↑ fibroblast proliferation)

↓

Cardiac dilatation & ↑ed After load

↓ further Systolic dysfunction


Lung capillaries
Pulmonary artery to lungs
Pulmonary vein to lungs
Vena cava from body
Aorta to body
Capillaries of body organs apart from the lungs

Diagnosis - 1) Symptoms : Shortness of breath that ⑤
wakes from sleep

2) Signs : Irregular Heart Sounds
Murmurs of Heart valves.
Clepts in lungs due to fluid in them
Cynosed fingers & toes
(low level of)

3) Chest xray → Fluid in lungs
Heart Enlarged

4) ECG → Inform Rythm & Size of Heart
Enlarged heart chambers

5) Echo : Any abnormalities in Heart valves,
Heart walls } are Enlarged
or
Chambers }

- Shows amount of blood in ventricles
i.e ventricular volume
- Amount of blood the ventricle pumps
with each beat.
i.e Ejection fraction

6) Cardiac catheterization : used to measure
Pressure in the heart - in each chamber

Treatment :

Dietary changes

1. To maintain wt
2. Reduce salt intake
3. Exercise - Walking
   Cycling
   Swimming

Life style changes

1. Restriction of smoking
2. Tobacco chewing
3. Used alcohol consumption

Medicines → To follow prescribed for Heart failure

1. Diuretics
2. Digitalis
3. Beta blockers
4. ACE inhibitors
5. ARB (Angiotensin receptor blockers)
6. Ca channel blockers.
7. Statins → To lowers cholesterol level. (LDL)

Surgery :

1. Clearing blocked coronary arteries by angioplasty or CABG.
2. Correction of abnormal Heart valves by replacing or Repairing Surgery
3. Heart transplant → with End stage HF

When all other treatments are not working

Prevention : 1) By Eating Healthy diet

2) Regular Exercise

3) High blood pressure → Pat's Regular BP checks

4) Obtaining Medical care for Coronary Artery disease

5) To Diagnose Early & Treated Early before they damage the Heart Muscle

Arteriosclerosis

Defination : Hardening of Normal flexible walls due to loss of elasticity of arterial ~~Muscles~~ Musculature.

ON the young stage the arteries are flexible & due to the presence of protein called elastin. There is loss of elastin causing thickening of the arterial walls. and artery becomes brittle

Involves small arteries whereas atherosclerosis involves Medium & large Vessels

Arteriosclerosis causes blood vessels to widen or rupture in aneurysm

~~Classification~~ :

There are 3 general patterns of different clinical and pathologic consequences.

1. Arteriosclerosis
2. Monckeberg Medial sclerosis
3. Atherosclerosis

As you know Arteriosclerosis affects small arteries and arterioles

HTN is associated c 2 forms of small vessel disease

1. Hyaline arteriosclerosis
2. Hyperplastic arteriosclerosis

shows luminal narrowing due to pink hyaline thickening in Arterioles.

Due to plasma protein leakage across injured Endothelial cells

↑ed smooth muscle cell Matrix synthesis in response to ch. Hemodynamic stress

Same lesions are common in Diabetic Microangiography

↓ when

Hyperglycemia induces Endothelial dysfunction

(Nephrosclerosis)

(3) Then hyaline arteriosclerosis causes diffuse impairment of renal blood supply & causes glomerular scarring.

2. Hyperplastic Arteriosclerosis : Occurs in in Malignant Hypertension

↓ Vessels Exhibit

Onion skin lesions — characterized by concentric laminated thickening of walls

Then lamination consist of smooth muscle cells & thickened & reduplicated basement membranes

2. Monckeberg Medial Sclerosis : characterized by calcific deposits in muscular arteries in persons older than 50 yrs of age.

Long elasticity of the walls of due to deposition calcification of Tunica Media.

3. Atherosclerosis → To continue main topic of Atherosclerosis

Atherosclerosis

Def: Is a disease in which plaque builds up inside arteries

↓

Made up of fat, cholesterol, Calcium & other substances found in blood

↓

Hardens & Narrows arteries

↓

Limit flow of O_2 rich blood to organs & Parts of body.

↓

supplying Heart: CAD / Heart attack

Brain: TIA / stroke

Arms & Legs: Peripheral arterial disease. Pain in legs while walking

Erectile dysfunction in men.

Etiology : Due to clogged vessels by cholesterol & fat

Pathogenesis : High levels of LDL
VLDL
cholesterol

① Infiltration into tunica intima

↓

Oxidised to form Ox-LDL

↓

Release Bioactive Phospholipids

↓

Activate Endothelial cells

↓

Express Leukocyte adhesion Molecules to adhere (Monocytes) leukocytes to intima & invade

↑

Causing

Adhering of Platelets

(Monocytes) Macrophages takes up large amount of lipids to form large foam cells in artery wall.

↓

- Immune sys gets activated locally & cause inflammation
- Then changes have negative effects
  1. Vase tone the ability to regulate the tone of vessel
  2. Inflammation ↑ scs
  3. Vessel wall prone to thrombosis
  4. Repair is inhibited

All these changes leads to Atherosclerotic lesions

Diagnosis

(1) Physical Examination:

1) Weak or absent pulse below narrowed area of affected artery

2) ↓ se BP in affected limb

3) whooshing sounds over arteries

4). Signs of pulsating bulge in affected area.

5). Evidence of poor healing where flow is restricted.

(2) Blood tests

1. High levels of cholesterol

(3) Doppler study: Helps to measure blood pressure along arm or leg → To know degree of any blockages & speed of flowing blood.

(4) Ankle - brachial index : BP of Ankle
Compared ē arm
Ak Abnormal difference indicate
Peripheral Vascular disease.

(5) ECG. Any abnormal wave
Report
- Previous Heart attack / if any which is in progress

(6) Angiogram : Dye shows narrowing or blockages.

Treatment-

1) Statin : Lower LDL level.
2) Antiplatelet drugs : Aspirin reduce sticking of platelets to narrowed vein form blood clot
3) Anticoagulants : Heparin or warfarin Helps in thinning of blood to prevent Clot formation

④ Anti hypertensives : β blockers
× ACE inhibitors
Ca channel blockers
} Slow the progression of Atherosclerosis

⑤ Surgery : Angioplasty : Mesh tube stent left in artery & helps by catheter / by laser technology removing the block.

Endarterectomy : Removal of plaque in the arteries.

Thrombolytic Therapy : Clot dissolving drug administered at the point of clot to break it.

Bypass surgery : CABG.

Prevention : Life style changes to prevent or slow the progression of atherosclerosis

Stop _____
Ex _____
Eat _____ fruits
_____ stress

Arrythmia

Def: Its abnormality of Heart's rhythm
where Heart palleein is too slow / too fast
Skip a beat / irregularly

Slow heart rate less than 60 beat/min " Bradycardia "
Fast rate > 100 beat/min " Tachycardia "

Arrythmias depend on site of origin i.e its also arrhythmia.

→ usually the heart beat arises at S.A node (Pace maker of Heart)
which is in Right atrium
from their sends impulse to AV atrioventricular node
↓
This in turn sends signal to ventricle to contract

→ Supraventricular arrythmias occur in upper areas of heart.
less serious than Ventricular arrythmias.

Ventricular tachycardia is fast irregular rhythm which can lead to Ventricular fibrillation — which is fast & irregular → beat so fast that its leading to sudden cardiac arrest stop pumping blood

Etiology : Heart diseases
Stress
Caffeine, Tobacco, alcohol, diet pills
Decongestants in cough & cold Medicines.

Pathophysiology : There are 2 types of cardiac tissue

1. ordinary myocardial muscle responsible for pumping action (i.e atrial & ventricular)

2. Specialized conducting tissue that generates Cardiac impulses & determine the order in which the muscle cells contract

So impulses are formed spontaneously is called autorhythmicity i.e SA, AV nodes.
SA node has highest frequency of spontaneous discharge : which is 70 times/min thus controls the contraction of heart

If SA node fails to function
↓
Next fastest node to takeover is AV Node
45 discharges/min
Next Purkinji fibres : 25/min

If there is any abnormality in mechanism impuls generated from centre in the nodes will cause arrythmias → called atrial fibrillation
or
flutter
or
Tachycardia

The orderly transmission of an electrical impulse through the conducting system may be related in an area of disease — Ischemia or previous MI

There an impuls travelling down Normal purkinje fibres fail to transmit impuls to adjacent fibres, & pass up to in reverse direction reexciting the cells may cause arrythmias

Diagnosis : Examination ⇒ Stethoscope
ECG
Electrophysiological studies : performed

to identify the origin of serious arrythmias cardiac catheterization done i Electrode on tip of catheter through arm or leg through vessels into heart which records & shows from where the arrythmia starts.

Treatment :→ Many arrythmias do not require treatment
→ For serious arrythmias : Treating known disease controls the arrythmia

→ Arrythmias → Treated By (1) Drugs — Beta blockers, quinidine, digitalis, Procainamide
In Emergency ← (2) Electrical shock
Cardioversion or defibrillation is used by applying Electrical shock to chest wall → which restores the heart to its normal rhythm followed by drug therapy to prevent arrythmias
(3) automatic implantable defibrillator
(4) Artificial Pacemakers
Corrects ventricular arrythmias which are life threatening to restore normal heart rhythm, More effective than drugs alone, Often used along & combination drugs.
sends Electrical signals to make the heart beat properly

(5) **Maze surgery** : Treats AF → where multiple incisions made in atrium to allow electrical impulses to move effectively. → Recommended to those not responded to Drugs or Cardioversion →

1. Define Hypertension, Discuss its causes & symptoms & Pathophysiology
2. Discuss the & treatment of Hypertension
3. Describe Ischemia & causes, symptoms & Management.
4. MI, its causes & its Management
5. Pathophysiology of MI & its Management.
6. Pathophysiology of Angina pectoris, causes symptoms & Treatment.
7. What is Heart failure, its causes & Management
   Rt sided and Lt sided failure & its Management.
8. Atherosclerosis & its pathophysiology
9. Arrhythmias

Question bank for CVS Chapter in Pathology

Pathophysiology.